In the Claims:

1. (Previously presented) A peptide having 2 to 10 amino acids, which is able to restore wild type function of human p53.

Claims 2-26. (canceled)

27. (Previously presented) The peptide of claim 1 having 3 to 7 amino acids.

28. (Previously presented) The peptide of claim 1 wherein the peptide incorporates the tri-peptide sequence WCT.

29. (Previously presented) The peptide of claim 28 wherein the peptide incorporates the pentapeptide sequence M-G/M/V-WCT.

30. (Previously presented) The peptide of claim 1 wherein the peptide has been modified at the C and/or N terminus to include a signaling or targeting moiety.

31. (Previously presented) The peptide of claim 30 wherein the signaling or targeting moiety is selected from the group comprising folate and the HIV Tat translocation sequence.

32. (Previously presented) A method of treating cancer comprising administering to a patient in need thereof a peptide having 2 to 10 amino acids which is able to restore wild type function of human p53.

33. (Previously presented) The method of claim 32 wherein the peptide has 3 to 7 amino acids.

34. (Previously presented) The method of claim 32 wherein the peptide incorporates the tri-peptide sequence WCT.

35. (Previously presented) The method of claim 34 wherein the peptide incorporates the pentapeptide sequence MG/M/V-WCT.

36. (Previously presented) The method of claim 32, wherein the peptide has been modified at the C and/or N terminus to include a signaling or targeting moiety.

37. (Previously presented) The method of claim 36 wherein the signaling or targeting moiety is selected from the group comprising folate and the HIV Tat translocation sequence.

38. (Currently amended) A method of screening a library of ~~molecules~~ peptides of 2 to 8 amino acids in length for the ability of members of the library to restore or modify the function of ~~a target protein~~ p53 in an intra-cellular environment, the method comprising:

(a) introducing ~~the~~ a library of peptides of 2 to 8 amino acids in length into host cells having a reporter system that allows for the identification of those cells in which the function of ~~the target protein~~ p53 has been restored or modified; and

(b) identifying those cells in which the function of p53 has been restored or modified.

39. Canceled

40. Canceled

41. (Currently amended) The method of claim 38 wherein the reporter system comprises a reporter gene which is operably linked to a sequence of nucleotides that provides a binding site for ~~the target protein~~ p53 or for a protein that associates with, or is a substrate for, ~~the target protein~~ p53.

42. (Previously presented) The method of claim 41 wherein the reporter gene is operably linked to a p21 or Bax promoter.

43. (Previously presented) The method of claim 41 wherein the protein product of the reporter gene includes a secretion signal peptide.

44. (Previously presented) The method of claim 41 wherein the protein product of the reporter gene includes a transmembrane domain.

45. (Previously presented) The method of claim 41 wherein the host cells have been transfected with the reporter gene.

46. (Canceled)

47. (Canceled)

48. (Currently amended) The method of claim ~~46~~ 38, wherein the library is introduced into the host cells in the form of nucleic acid constructs which encode the peptide library.

49. (Currently amended) The method of claim ~~46~~ 38, wherein each member of the peptide library has the sequence M-G/M/V-(X)n, wherein n is an integer from 3 to ~~18~~ 6, M is methionine, G is glycine, V is valine and each X, which may be the same or different, is any genetically coded amino acid.

50. (Currently amended) The method of claim 38, wherein the ~~molecular~~ peptide library has at least 500 different members.

51. (Previously presented) The method of claim 38 wherein the host cells are eukaryotic cells.

52. (Previously presented) A pharmaceutical composition comprising a compound, identified by a method according to claim 38, which is able to restore

wild-type function to a mutant protein.

53. (Previously presented) A method of treatment, wherein the condition is selected from cancer, cystic fibrosis, sickle cell anemia, phenylketonuria, multiple carboxylase deficiency, methylpurine DNA glycosylasedeficiency (MPG), ataxia and chemotherapy resistance due to mutations in the gene coding for methylguanine-DNA methyl transferase (MGMT), comprising administering to a patient in need thereof the pharmaceutical composition of claim 52.

54. (New) A method of identifying a peptide of 2 to 8 amino acids in length having the ability to restore or modify the function of p53 in an intra-cellular environment comprising:

(a) introducing a library comprising nucleic acid constructs encoding peptides of 2 to 8 amino acids in length into host cells having a reporter system that allows for the identification of those cells in which the function of p53 has been restored or modified;

(b) identifying a cell in which the function of p53 has been restored or modified; and

(c) identifying the peptide in the cell of step (b).

55. (New) A method of identifying a peptide of 2 to 8 amino acids in length having the ability to restore or modify the function of p53 in an intra-cellular environment comprising:

(a) introducing a library comprising peptides of 2 to 8 amino acids in length into host cells having a reporter system that allows for the identification of those cells in which the function of p53 has been restored

or modified;

(b) identifying a cell in which the function of p53 has been restored or modified; and

(c) identifying the peptide in the cell of step (b).